The Newsletter of the Australian and New Zealand Society of Paediatric Dentistry

Synopses

DECEMBER 2000, ISSUE 23

Approaching the Question of Microvascular Remodeling

*Gue S, *Cameron A, +Zoellner H
*Dept. of Paediatric Dentistry, Westmead Dental Clinical School, Westmead Hospital
+Dept. of Oral Pathology, Faculty of Dentistry, The University of Sydney, Westmead Hospital Dental Clinical School

Dr Sam Gue was the winner of the 2000 Colgate Oral Care Paediatric Dentistry Graduate Student Award. Address Correspondence to: Dr. Sam Gue, Paediatric Dentistry Registrar Paediatric Dental Department Westmead Hospital and New Children's Hospital Darcy Road, Westmead 2145 University of Sydney, Sydney, New South Wales

Microvascular Remodeling as a Model for Pattern Formation

The microcirculation serves to supply nutrients to the tissues as well as to remove waste products and this principal role demands the formation of a complex branching structure by the constituent endothelial cells. Although numerous signals have been identified as important for microvascular development including Fibroblast Growth Factor, Vascular Endothelial Growth Factor, Angiogenins and Ephrins, significant problems remain to be solved in understanding microvascular morphogenesis. In particular, it is unclear what mechanisms are responsible for organization of individual endothelial cells into complex microvascular networks. The difficulty of this problem becomes apparent when it is considered that microvascular flow in each part of the microcirculation is functionally dependent upon flow in all of the remaining parts, while individual endothelial cells are only in contact with adjacent cells and so have no direct opportunity to communicate with their more distant but nonetheless functionally dependent fellows. One approach to dealing with this problem is to study microvascular networks undergoing remodeling.

Endothelial Apoptosis in Microvascular Remodeling

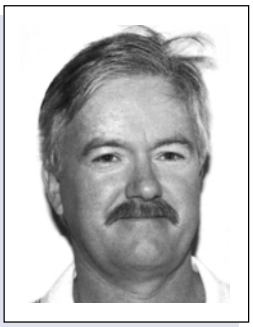
During wound healing, highly vascular granulation tissue undergoes significant microvascular remodeling and maturation to become mature scar tissue. The dramatic extent to which excess vessels are removed during wound maturation is illustrated by the change in color from granulation tissue which is pink with new vessels to white scar tissue containing very few blood vessels. This process is mediated by apoptosis of the constituent endothelium while endothelial cell apoptosis has also been shown to be responsible for microvascular remodeling during pressure induced atrophy of the parotid and involution of the mammary gland. In addition to this, endothelial cell apoptosis has been found to contribute to delayed wound healing in diabetes, hypertensive vasculopathy and scleroderma. From this, it appears that endothelial cell apoptosis plays a central role in both physiological and pathological microvascular remodeling and it is likely that this is also important during orthodontic tooth movement and bone remodeling.

Apoptosis: A Matter of Life and Death

Apoptosis is currently accepted as the principal mechanism whereby excess cells are removed from the body. Morphologically and ultrastructurally, apoptosis is recognized by cellular fragmentation into apoptotic bodies often containing fragments of condensed nuclear material but with intact organellar structures. endothelial cell apoptosis appear to be ultrastructurally unique in that complex canalicular structures form in apoptotic particles. This has been proposed as a mechanism aiding the mechanical fragmentation of apoptotic endothelial cells and thus minimizing the micro-embolic potential of these cellular fragments. Internucleosomal cleavage of DNA into 180 base pair fragments is a widely accepted biochemical marker for apoptosis. This contrasts with necrosis, in which homeostasis is lost due to toxic or other noxious agents. In necrosis there is disruption of membranous structures, degeneration of mitochondria and ribosomal detachment from rough endoplasmic reticulum. In addition to this, DNA fragmentation in necrosis is random so DNA has a smeared appearance in gel electrophoresis. Further, since necrosis is due to tissue damage it usually occurs in large groups of cells while apoptosis is described as occurring in single cells. From this, apoptosis appears to be tightly controlled at the level of individual cells rather than in a tissue wide manner.

Continued on Page 3...

this issue



President's Report

PROFESSIONAL RESPONSIBILITY AND PATIENT WELFARE

My earliest recollections of children's dentistry were occasional visits to a mobile government dental clinic in a railway carriage at ten years of age. The clinic visited country Queensland on a regular annual run with the itinerant dentist. To my best knowledge, I had amalgam fillings done in my primary teeth and I can recall two distinct smells; one I think was eugenol and the other may have been formocresol. I can vividly remember being asked to mix the mercury with the silver filings in a small bowl for the dentist, following the struggle of slow speed cavity preparation without a local anaesthetic.

What was happening in the world of dentistry for children in the 1960's? The only available written documentation was the *Journal of Dentistry for Children*. At the time, it was the only dedicated journal to children's dentistry and a quick scan of the content of four issues suggests that the problems in the 1960's continue to be similar to those problems that children's dentists face today. For example, the journal issues included articles on general anaesthesia, a technique on vital pulpotomy on young permanent teeth, dental treatment for children with congenital heart disease, caries in hospitalised children, the Howe technique for pit and fissure caries, concern over the availability of dental services for children and more interestingly, dental care from birth to two years of age. After reviewing the journal, I considered what had changed over the last 35 years.

When I entered dental school in 1972, I realised I was not the only student that had a lot of cavities as a youngster. In fact some of those fillings still remain and are working just a well, despite the concerted attempts from my dental student colleagues to replace them. Restoration in 1972 was limited to amalgam in the posterior teeth and silicate cement in the anterior teeth. Seldom were children younger than five years of age treated (except for emergency extractions) and the benefit of fluoride was only just beginning to emerge. When I finished my speciality training in 1991, dental decay in children was certainly on the wane. Systemic and topical fluorides had decreased the prevalence of smooth surface caries and I discovered alternatives to amalgam including composite resins, glass ionomer cements and stainless steel crowns. Most importantly, there was a professional realisation that the age for a child's first dental visit should be much younger than five years.

After a few years in public hospital practice and part time academics, it dawned on me that there must be a better approach to maintaining children's oral heath than restoring decayed teeth. The surgical approach to disease management has since been outdated by the medical model of disease control and prevention. I am convinced that dentists that treat children have a distinct advantage over all other disciplines of dentistry. Only we can reduce the impact of dental caries and it's effect on oral and somatic development through early intervention and initiation of a comprehensive preventive program in high-risk children.

Although dental decay in children will never be eradicated, the treatment options for disease management are extensive, including effective antimicrobial and fluoride preparations. Most children in Australia and New Zealand drink fluoridated water and almost all paediatric toothpaste contains fluoride. Fissure sealants reduce occlusal caries and there are hundreds of toothbrush designs to effectively remove dental plaque. More importantly, increasing numbers of parents, doctors and dentists recognise the importance of early examination and guidance of the developing primary dentition and its importance for general growth and development. In addition to the *Journal of Dentistry for Children*, there are a number of journals that publish contemporary issues in paediatric dentistry.

How about the next 35 years? Discussion on pulp treatment in the primary and permanent dentition will continue, probably in the area of induced osteogenic repair. With new medical therapies to treat childhood diseases that were once fatal, I expect there will be more complex issues in managing children with special health care needs. The genes responsible for tooth development will be mapped and a host of diagnostic tests will be available to identify children at highest risk for dental disease. Gene replacement therapy may be utilised for disease prevention, subject to public acceptance. New techniques for cavity debridement and restoration placement will evolve. Infants and pre-schooler attendance will increase due to the perceived importance of early identification and intervention. The presence of child friendly techniques will improve both compliance and behaviour management during the active treatment phase.

Paediatric dentistry is an exciting and energetic field of dentistry but is not without its share of controversy. However, it is our professional responsibility to embrace those concepts and techniques that further our patient's health and welfare. ANZSPD will continue to promote the practice and science of paediatric dentistry through its organisational activities. Even though the challenge to continue is great, the creativity, energy and intellect of future members will allow the society to progress and develop.

Kerrod B Hallett

an invitation

TO ALL ANZSPD MEMBERS

An invitation is extended to all members of the Australian and New Zealand Society of Paediatric Dentistry (ANZSPD), to participate in the development of "Oral Health Care Policies and Standards of Care Guidelines for Paediatric Dentistry", which is currently being produced by the Australasian Academy of Paediatric Dentistry (AAPD).

A full day meeting will be held at Rydges Southbank Hotel, Brisbane, Queensland (ADA Congress Hotel) on Thursday 3 May 2000, just prior to the 30th Australian Dental Congress. Further details and copies of the proposed protocols may be obtained by contacting Ms Sarah Raphael, Secretary, AAPD

Email: ks.mekertichian@bigpond.com
Address: 151 Woodland Street
Balgowlah NSW 2093
Telephone: (02) 9907 8123.

Peter J Gregory, President
Australasian Academy of Paediatric Dentistry

...Continued from Page 1

Co-ordination of Endothelial Apoptosis: A Significant Problem

If, however, apoptosis is regulated at the level of individual cells, additional problems become apparent in regulating apoptosis of endothelial cells during vascular remodeling. It is clear that if endothelial cell apoptosis is not properly coordinated, the microvascular network will be disrupted resulting in either ischaemic necrosis or bleeding. Since this does not occur in-vivo, it is clear that there must be mechanisms to co-ordinate endothelial cell apoptosis and maintain vascular perfusion during micro-vascular remodeling.

Since the principal function of blood vessels is to carry blood, it is possible that blood flow provides a mechanism linking microvascular function to regulation of endothelial cell apoptosis. Supporting this possibility is the observation that microvessels carrying blood are maintained during microvascular remodeling in-vivo, while those vessels which are poorly perfused or have oscillatory flow undergo degeneration. An important conclusion from this is that the default status of endothelial cells appears to be apoptosis, with endothelial cells only surviving if receiving survival signals from flowing blood. This model is illustrated in Fig. 1, where vessels having indifferent or conflicting flow in the "initial vasculature", are removed by endothelial apoptosis to produce an optimized "final vasculature".

Chemical Plasma Factors and Laminar Shear Stress Inhibit Endothelial Apoptosis

The two types of anti-apoptotic signal which might act upon endothelial cells in functional blood vessels are shear stress or chemical plasma factors. Laminar shear stress has been shown to inhibit apoptosis in isolated cultured endothelial cells and it is likely that this plays some role in-vivo. However, since isolated endothelial cells grow and survive in the absence of shear stress, it seems that although shear may inhibit endothelial cell apoptosis, it is not a critical anti-apoptotic factor for these cells. In contrast, serum deprivation causes rapid endothelial cell apoptosis in the absence of shear, suggesting that chemical plasma factors are critical anti-apoptotic signals for endothelial cells. This laboratory has been investigating this possibility and we have found that serum contains potent anti-apoptotic factors for endothelium, with at least one of these factors being serum albumin. Although most effort has been directed towards understanding the way in which serum albumin may mediate this anti-apoptotic activity, we have more recently revisited the question of the possible effect of shear stress upon endothelial cell apoptosis.

A Simple Rocking Platform Model to Stimulate Endothelium with Shear Stress

Experiments stimulating cells with shear stress are quite difficult, primarily because highly specialized and complex apparatus is required. This makes performing preliminary experiments problematic, as significant expenditure on new equipment can not always be justified without any evidence that the new line of investigation might be fruitful. To get around this rather practical problem, we established a very simple experimental system, in which endothelial cells grown to confluence in tissue culture flasks were simply rocked back and forth on a laboratory rocker. In this way, the culture medium overlying the cells was allowed to wash forwards and backwards over the cells, thus exposing the cells to shear stress. By aligning tissue culture plates in parallel with each other, as well as being careful to load equivalent amounts of culture medium in each flask, it was possible to stimulate sets of three or more flasks as triplicates to facilitate statistical analysis. Cells in these "rocking flasks" could then be compared with parallel cultures treated in the same way in the same incubator, but on a stable incubator shelf. We knew from earlier work that endothelial cells become more rapidly apoptotic in serum free conditions while albumin inhibits this process. Also, we had shown that endothelial cells do not proliferate in flasks unless stimulated with specific growth factors prepared from bovine hypothalamus. Because of this, we performed these experiments with both "rocking" and "static" cultures with three types of culture medium: i) medium in which endothelium has very low levels of apoptosis (M199 with 20% serum); ii) medium in which endothelum is slightly more apoptotic (M199 with 4% serum albumin); and iii) medium inducing high levels of endothelial apoptosis (M199 alone). Also, earlier work had shown that because endothelium does not proliferate in these conditions, it is possible to quantitate the extent of endothelial apoptosis by simply counting the number of cells surviving experiments. These were are easy to separate from apoptotic cells because endothelial cells detach early during apoptosis and can be washed away, while surviving cells are all adherent to the culture surface.

> "Instead of finding that shear exposed cells were protected from apoptosis, what we found was that fewer cells survived on the rocking platform than in static culture. This was the absolute opposite of what was expected."

Data Were the Opposite to What Was Expected

Instead of finding that shear exposed cells were protected from apoptosis, what we found was that fewer cells survived on the rocking platform than in static culture. This was the absolute opposite of what was expected, as we had argued that functional signals should inhibit endothelial apoptosis and shear stress was clearly a functional signal. Not only that, but while performing these experiments, other workers had published work with laminar flow chambers showing reduced endothelial apoptosis in the presence of laminar shear stress. At first, we rejected our data, thinking that "something had gone wrong". However, the more experiments were repeated, the more clear it became that endothelial cells in our rocking platform model die more quickly than parallel cells sitting on the incubator shelf. This difference was seen regardless of the culture medium used, although the gap between survival and death seemed most pronounced when cells were cultured in serum free conditions with albumin.

It became important to check that cells were actually apoptotic and not simply necrotic. To look at this, we examined the cells by transmission electron microscopy, DNA gel electrophoresis and FACS analysis and have found that the cells are clearly apoptotic. The only conclusion left for us was that we were inducing endothelial apoptosis by culture on the rocking platform.

Does Oscillating Shear Stress Affect Endothelial Apoptosis Differently to Laminar Shear Stress?

As mentioned above, other workers had shown that laminar shear stress protects endothelium from apoptosis so that our task became one of trying to reconcile our observations with those of these other workers. Comparing our work with that reported in the literature, the most obvious difference was that we had stimulated cells with oscillating shear stress while our colleagues had stimulated their cells with continuous laminar shear stress. Also, we knew that increasing the rate of rocking in our platform rocker model increased the extent of apoptosis. From this, we concluded that oscillating shear stress induces apoptosis while laminar shear stress inhibits this.

Continued on Page 4...

...Continued from Page 3

The Possible Biological Relevance of Oscillating Shear Stress

It is always difficult to meaningfully extrapolate observations with isolated cultured cells to the in-vivo situation. Cultured endothelial cells grown as a monolayer on a plastic plate are clearly a very poor model for endothelial cells in actual micro-vessels. Nonetheless, it seems fair to assume that cells carry at least some of their in-vivo properties across into cell culture, and that the behavior of cultured endothelium usually reflects some aspect of the real biology we wish to study. Accepting the limitations of cell culture models, but on the assumption that their behavior reflects some aspect of biology, it became important to try and imagine some way in which oscillating shear induced endothelial apoptosis might be biologically important.

In-vivo observations by other workers have infact revealed that in remodeling apoptotic vessels, where flow is already impaired, the blood often oscillates back and forth. Further apoptosis in these vessels eventually results in removal of these microvascular limbs and it seems reasonable to suggest that the increased apoptosis in "rocking" cultures reflects a mechanism for accelerating endothelial apoptosis in these sorts of degenerating blood vessels. Similar events may occur in vessels where there are flow conflicts (Figure 1). Another possibility, is that the oscillatory shear stress generated on the rocking platform mimics the oscillations in flow or turbulence generated when vessels become excessively tortuous. In this way, apoptosis of endothelium in such excessively tortuous vessels might help to optimize microvascular shape.

If this is the case, it becomes interesting to speculate as to the possible ways in which this mechanism might be inhibited in conditions where vessels become increasingly tortuous, such as chronic periodontitis. Perhaps inflammatory cytokines inhibit this process? Before extending work to these sorts of questions, however, it is more important to properly characterize the current observations with oscillating shear.

The Need for an Improved Experimental Model

As mentioned above, the rocking platform model was used primarily as a simple and inexpensive way of stimulating endothelium with shear stress. Although permitting experiments to be performed, it was not possible to directly compare the response of these cells with others exposed to true, uniform, unidirectional laminar shear stress. Also, because the flow varies in direction and velocity according to the angle of the rocking platform, it was not possible to properly quantitate the levels of shear used. Worse still, turbulence generated at the extremes of each cycle makes calculation of shear stress impossible. Since we now have this interesting data, further expenditure on more complex and expensive equipment seems justified. To this end, we are currently assembling an apparatus which will allow us to study these phenomena more carefully. Despite numerous technical difficulties and setbacks, we hope to be able to initiate experiments with this much more sophisticated model system in the near future.

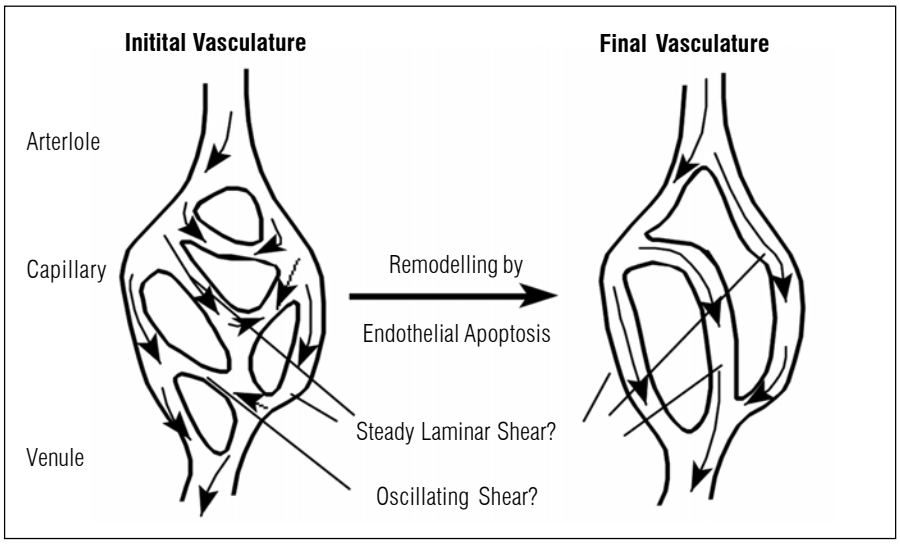


Fig. 1. Illustration of the effect of blood flow in individual micro-vessels upon survival of vessel segments during microvascular remodeling. Blood enters the microvasculature via arterioles, passes through capillaries and exits via venules. Blood flow is indicated by arrows and vessel segments where blood flow is poor are lost by endothelial apoptosis with the effect that microvascular structure is optimized. Poor flow may result from flow conflicts or insufficient arterial supply relative to the dependent capillary bed. As discussed in the text of this article, we have found that chemical plasma factors have a central role in signaling the presence of flowing blood in functional blood vessels, inhibiting apoptosis and so helping to control the structure of the microcirculation. Other workers have demonstrated a protective effect of steady laminar shear stress while we find that that oscillating shear seems to stimulate endothelial apoptosis. This may contribute to removal of vessel segments where there are flow conflicts as well as excessive tortuosity.

Dental Project for Cambodia
2001-2002

Since the 1980s, progress has been made in re-establishing oral health services and training in Cambodia following two decades of war, political instability and hardship. The Faculty of Dentistry in Phnom Penh has received some help in recent years from non-governmental organisations, and a few Cambodian dentists have had the chance to make short educational tours overseas to improve their knowledge. Unfortunately only 4 Cambodian dentists have had the opportunity to study abroad. Of these one has since retired and 2 have gone to live in the US. In order for Cambodia to continue to improve oral health services for its people, upgrading the knowledge and skills of staff at the Faculty of Dentistry must be a priority.

The idea for this project came about during the Cambodian Dental Association Conference in January 2000, when a group of foreign dentists who attended the meeting formed a group called "*Friends of the Cambodian Dental Association*". Following discussion with the Cambodian dentists the group proposed to help upgrade the knowledge and skills of the teachers at the Faculty of Dentistry by running 2-year part-time diploma courses in each of the clinical disciplines. The *first* of the courses to be run is a *Diploma of Paediatric Dentistry* and will begin in January 2001.

The course for 4-5 Cambodian dentists has been developed with Cambodia's specific needs in mind, and will involve intermittent visits by up to 8 overseas volunteer paediatric dentists, mainly from Australia and NZ. Each visiting lecturer will teach part of the course as well as providing clinical supervision. The period of each visit will be 1-2 weeks with about 2 months between visits during which time students will continue to treat children on their own, and carry out prescribed readings and assignments. Those Cambodian dentists invited to attend the course include teachers at the Faculty of Dentistry and the Dental Nurses training school, and one or two general dentists who have a particular interest in or responsibility for treating children. The local coordinator for the course is Dr Poum Sen, Lecturer in Paediatric Dentistry at the Faculty of Dentistry in Phnom Penh.

Along with the teaching, we intend to upgrade the sparsely furnished paediatric clinic at the Faculty of Dentistry, by seeking donations of supplies, instruments and equipment. If this first course in paediatric dentistry is successful, it is intended that courses in other clinical disciplines will be introduced later.

Anyone interested in helping support this project should contact:

Callum Durward
Paediatric Dentist
Greenlane Hospital
Private Bag 92-189
Auckland New Zealand
E-mail: c.durward@clear.net.nz

Oral Manifestations of Oncology Treatment in Paediatric Patients

Peter Readman, Paedodontic Registrar Princess Margaret Hospital
Supervisor: Robert Bower, Consultant Periodontist Princess Margaret Hospital

Inroduction

Malignant disease ranks only second to accidents as the most common cause of death in children under 15 years of age.[1] The most commonly diagnosed paediatric malignancies are leukaemias, followed by tumours in the central nervous system, lymph nodes, bones, joints and cartilage, and those of endocrine origin.[2]

Complications resulting from neoplasia, or secondary to cancer treatment, frequently manifest in the oral cavity.[3,4] Surgery, radiation and cytotoxic chemotherapy disturb the normal integrity of the mouth both anatomically, histologically and physiologically. This paper will review the oral complications of both childhood neoplasia and cancer therapy.

Oral Complications of Systemic Cytotoxic Chemotherapy

Cancer chemotherapy protocols are designed to obliterate malignant cells at the primary lesion and elsewhere throughout the body.[6] In acute leukaemia, the drug schedules are targeted primarily against bone marrow, inducing a pancytopenia.[6] The drug-induced myeloimmunosuppression correlates closely with vulnerability to infection. Cytotoxic chemotherapeutic agents may therefore produce adverse side effects by direct toxicity at the cellular level or indirectly, characterised by drug-induced pancytopenia.[6]

About 40 to 50 per cent of all patients receiving chemotherapy develop oral changes, however, these figures approach 90 per cent in children.[1,5,7-9] The increased mitotic index in children and associated rapid epithelial turnover and maturation rates is thought to be responsible for their greater susceptibility to cancer therapy.[9]

A longitudinal study by Fayle and Curzon investigated the oral complications of 43 paediatric oncology patients aged two to 14 years receiving treatment for malignant disease.[3] Forty (93 per cent) developed oral complications during the study period which emphasizes the need for dental involvement in paediatric oncology patients.

The nature and incidence of oral problems observed during treatment are illustrated in Table 1.

Mucositis and Ulceration

The oral mucosa is comprised of cells with a high mitotic index, making these tissues particularly vulnerable to the direct effects of cytotoxic chemotherapy.[1,10] By inhibiting cell growth, cytotoxic chemotherapy alters the integrity of the oropharyngeal mucosa, thus creating a portal of entry for the microbial flora found within the oral cavity.[1,10] In addition, a gross reduction in granulocyte numbers, as well as modifications to their functional capabilities (eg. reduction in migration, phagocytosis, and antibacterial effect), leads to an increase in susceptibility to infection.[1,10,11]

Barrett proposed that five interrelated factors contribute to the clinical breakdown and ulceration of the oral mucosa following cytotoxic chemotherapy.[12] First, the relatively rapid rate of cell turnover in the oral mucosa is important in explaining its susceptibility. Second, degenerative changes in the supportive submucosal collagen may play a key role in the development of ulceration following cytotoxic chemotherapy. Third, the structural characteristics of the mucosa and submucosa in areas of keratinised and nonkeratinised mucosa may help explain their apparent differing susceptibilities. Nonkeratinised mucosa generally contains a loose connective lamina propria, which offers little support and facilitates mobility of the overlying epithelium. In contrast, keratinised mucosa is firmly bound to periosteum by a collagen rich lamina propria. Fourth, regional susceptibility may also be explained by the presence or absence of keratin, which offers physical protection to the epithelium. Fifth, neutropenia and local secondary infection may progress to overt ulceration as a result of the patients impaired healing response.[12]

Mucositis, ulceration and erosion are the most common oral complications of cancer treatment.[1,3,13,14] Initial symptoms include a burning sensation, dryness of the mouth, tingling of the lips and pain.[1] Depending on the chemotherapeutic regimen used, erythematous mucositis develops in three to five days after the initiation of therapy and a localised or generalised ulcerative mucositis in seven to ten days.[3,5,13] These painful ulcerative lesions are noted primarily on nonkeratinised tissues, such as the buccal and labial mucosa, the ventral and lateral surfaces of the tongue and the floor of the mouth. Keratinised tissues such as the hard palate, attached gingivae and dorsal surface of the tongue are rarely subject to mucositis but may become painful.[3,5,13] Predicting which patients will suffer mucositis remains difficult as the capacity to tolerate cytotoxic chemotherapy varies greatly between individuals.[12,15] In almost all cases, patients involved have low neutrophil counts (less than 0.5 x 10^9/L).[3] The ulcers usually last five to ten days with healing coinciding with an improvement in the neutrophil count.[3]

Cytotoxic chemotherapeutic agents associated with mucositis include antimetabolites and in particular methotrexate, cytarabine and 5-fluorouracil, and antitumour antibiotics such as daunorubicin, doxorubicin, bleomycin and hydroxyurea.[5] If cytotoxic chemotherapy is given in combination with total body irradiation prior to bone marrow transplantation a rapid-onset mucositis may develop.[1,5]

Treatment of mucositis is primarily palliative aimed at minimising mucosal trauma and improving patient comfort.[13] During radiation and chemotherapy, the oral tissues should be kept moist by frequent water rinses.[5] If mucous is thick

ORAL PROBLEM	NUMBER AFFECTED (N=43)
Mucosal Ulcers	28 (65%)
Lip Cracking	15 (35%)
Lymphadenopathy	15 (35%)
Mucositis/Erythema	13 (30%)
Oral Bleeding	9 (21%)
Severe Gingivitis	8 (19%)
Herpes Simplex	6 (14%)
Candidiasis	5 (12%)
Dry Mouth	2 (5%)

Table 1. Incidence of oral problems observed during treatment[3]

Continued on Page 6...

...Continued from Page 5

it can be diluted by rinsing with a five per cent solution of sodium bicarbonate.[5] Rinsing the mouth after brushing with a salt and soda (half a teaspoon salt and one teaspoon baking soda in one pint of water) is recommended.[14] Saline solutions are thought to aid in formation of granulation tissue and promote healing, while sodium bicarbonate dissolves mucin and loosens debris.[14]

To prevent oral mucositis, patients undergoing cytotoxic chemotherapy usually receive some form of prophylactic mouthwash.[6] The double blind, randomised clinical trials of Ferretti et. al., and Rutkauskas et. al., support prophylactic use of chlorhexidine in immunocompromised patients.[7,16] These studies showed a significant reduction in both incidence and severity of mucositis in chlorhexidine treated patients.[7,16] Consequently, patients undergoing cytotoxic treatment may achieve better nutrition, fewer septic episodes, and shorter hospital stays.

Benzydamine containing mouthwash (Difflam) is often used in the treatment of mucositis.[17] Benzydamine hydrochloride is a topical nonsteroidal anti-inflammatory drug with analgesic, antipyretic, antimicrobial and local anaesthetic activity. When applied topically as an oral rinse it offers the unique property of concentrating in inflamed tissues. Benzydamine is used widely in the management of pharyngitis, radiation mucositis, aphthous stomatitis and post-operatively following dental surgery. This mouthwash is also available with the addition of chlorhexidine (Difflam-C) improving its antimicrobial properties.[17]

Kamillosan is another mouth wash which has been recently reviewed in the dental literature. Research has been aimed towards developing an irrigation solution that will prevent or reduce mucositis and promote epithelialization of desquamated regions of the oral mucosa.[18] Kamillosan solution is prepared from the flower of the camomile plant. The main constituents are chamazulene, levomenol, polyins, and flavinoids. These constituents are antiinflammatory and spasmolytic, and promote granulation and epithelialization through a favourable effect on the energy-dependant process of cell metabolism. Carl et. al., studied the prophylactic and therapeutic use of Kamillosan oral rinse in 20 patients who received head and neck irradiation and 78 patients who received systemic chemotherapy reporting a reduction in both intensity, onset and duration of mucositis.[18]

1. Bacterial Infections

Bacterial infections are a significant cause of morbidity and mortality in myeloimmunosupressed patients.[13,18] The majority of oral bacterial infections in children involve secondary involvement of mucosal ulcerations due to therapy-related neutropenia.[13,18] A wide range of bacteria, including odontopathic, periodontopathic, and transient pathogens of the oral cavity may infect ulcerative lesions.[13] Predisposing factors to infection include, neutropenia, mucosal damage, periodontal disease, dentoalveolar abscess, mobile deciduous teeth, cellular immune dysfunction, haematologic immune dysfunction and iatrogenic procedures.[5]

The clinical appearance of potentially severe local infections can be reduced or even absent during chemotherapy as a result of induced neutropenia.[1,6,10,19] This particularly applies to gingival and periodontal inflammation.[20]

> "The incidence of oral infection is reduced when all definitive dental treatment is completed before the commencement of cytotoxic chemotherapy."

The incidence of oral infection is reduced when all definitive dental treatment is completed before the commencement of cytotoxic chemotherapy.[1] Patients who receive cytotoxic chemotherapy should have a thorough oral examination including both clinical and radiographic examination.[13] If therapy has already commenced, it is still recommended that patients be evaluated as early as possible to establish a baseline examination which will assist in the monitoring and treatment of oral complications.[13]

Optimal dental treatment should include a prophylaxis, restoration of carious teeth, replacement of unsatisfactory restorations, smoothing of fractured enamel or rough restorations, extraction of necrotic, infected or exfoliating teeth and elimination of prostheses.[13] Good plaque control before chemotherapy is essential to reduce the morbidity associated with soft tissue infection.[1,13]

Oral hygiene procedures, both mechanical debridement and mouth rinses, should be performed three times a day.[5] If tissue complications develop or deteriorate, the frequency of oral care should increase appropriately.[5] A number of anaesthetics, analgesics and mucosal coating agents are available for the control of pain. They include Benadril, Kaopectate, milk of magnesia, Orabase, viscous xylocaine, Cepacaine, Difflam mouth gel, Oratect Gel and systemic analgesics.[5] There is no experimental evidence, however, to establish the efficiency of any one or combination of these agents in the management of pain.[5]

Conventional tooth brushing during myeloimmunosuppression may increase the risk of bleeding and infection.[13] A modified mechanical approach is recommended consisting of either irrigation with a Water Pik or cleaning with a disposable sponge, piece of gauze or cotton swab with a topical antimicrobial agent.[13]

Patients receiving long-term chemotherapy may experience subtle changes in their saliva from microbial shifts to a decrease in the quality and quantity of saliva.[5] Brown et. al., reported a higher concentration of *Candida sp* and *Staphylococcus sp* in immunodeficient patients. However, a lower recovery of *S mutans* and a lower caries experience in immunodeficient patients was noted.[21] These findings indicate that other protective mechanisms may compensate for immunoglobulin deficiency.[21]

2. Fungal Infections

Fungal infections in patients undergoing cytotoxic chemotherapy most commonly involve *Candida albicans*.[13] Other less common fungal infections in immunosuppressed patients involve *Histoplasma capsulatum* and *Cryptococcus neoformans*. Under normal conditions in the oral cavity, the growth of *C. albicans* is inhibited by commensal microorganisms and by an intact immune system. Once pathogenic, *C. albicans* may spread to the oesophagus or trachea via deglutition or droplet aspiration, or via haematogenous spread. The most serious infections involve major organ involvement or disseminated candidiasis.[13]

The incidence of candidiasis in paediatric patients receiving chemotherapy has been reported at 15 and 30 per cent.[3,22] Candidiasis in children occurs most frequently on the dorsum and lateral surfaces of the tongue, the commissures of the lips, and the buccal, gingival and palatal mucosa.[1,6] Candida colonisation appears as raised, creamy white, curd-like strands, beads, flecks and patches that adhere to the underlying mucosa and leave painful shallow ulcers when removed.[6] Patients receiving broad spectrum antibiotics or steroids, may have atrophic candidiasis which presents as a red often painful tongue.[1]

Clinical management of oral candidiasis in children is similar to that in adults and consists of antifungal agents.[1] Nystatin oral suspension or amphotericin B syrup or troches are often given after early diagnosis of oral candidiasis.[6] These agents

can be cariogenic as a result of the high sugar content and must therefore be accompanied by excellent oral hygiene in paediatric patients.[1]

Epstein et. al., assessed the potential role of prophylactic mouthwashes in the reduction of oral complications in patients receiving chemotherapy for leukaemia.[23] This study, along with many other publications, did not identify any prophylactic effects in Candida colonisation, clinical oropharyngeal infection or fungaemia with the use of nystatin. Other studies of Candida prophylaxis using nystatin have often been based on small patient series which have produced inconsistent results.[23-26,27-31]

Compliance with nystatin rinsing is often limited by taste complaints, nausea and vomiting. Recent research has therefore focussed on fluconazole in Candida prophylaxis. Bodey et. al., studied oropharyngeal candidiasis in 112 adults undergoing therapy for metastatic malignancies.[32] In this double-blind study only two per cent of patients receiving fluconazole developed oropharyngeal candidiasis compared to 28% for those receiving placebo. Of the patients receiving placebo, those who also received antibiotic therapy or systemic adrenal corticosteroid therapy suffered a higher incidence of oropharyngeal candidiasis at 30 per cent and 44 per cent respectively. Candidiasis was also evident in 54 per cent of placebo patients who were colonized by Candida sp at the commencement of the study. Fluconazole provided effective prophylactic cover for oropharyngeal candidiasis in this study with few reported side effects.[32]

Quirk et. al., retrospectively studied 196 patients who underwent bone marrow transplantation.[33] Patients were divided into two study groups; 1) those receiving oral nystatin/amphotericin or 2) those receiving oral fluconazole. Fluconazole significantly reduce the incidence and severity of oropharyngeal candidiasis, showed good compliance and had minimal side effects. Oropharyngeal candidiasis occurred in 54 percent of those patients in the nystatin/amphotericin group and in only seven per cent of those patients receiving fluconazole.[33]

The prophylactic use 0.12 percent chlorhexidine mouthrinse was shown by Ferretti et. al., to reduce the incidence of oral candidiasis in patients undergoing intense chemoradiotherapy after bone marrow transplant.[16] In a prospective, randomised, double-blind study, patients were assigned mouthrinses containing either 0.12 per cent chlorhexidine or a control mouthrinse identical in composition but without chlorhexidine. There was an absence of clinically evident oral candidiasis in the chlorhexidine-treated group compared with 64.7 per cent cumulative incidence of oral candidiasis in the control group.[16] Furthermore, two deaths from Candida sepsis occurred in the control group during the early post-transplant period.[16]

Nystatin and chlorhexidine should not be used simultaneously. Combining these agents results in the formation of a chlorhexidine-nystatin complex that appears to be ineffective against candida.[34] If the fungal infection becomes disseminated, fluconazole or ketoconazole tablets may be effective.[6] Intravenous amphotericin B however, is the preferred drug for treating serious systemic infections.[6]

3. Viral Infections

Herpes simplex virus (HSV) infection is the most common viral infection associated with cancer treatment.[1] The virus may occur as a primary infection, or more commonly as reactivation of the latent virus during periods of immunosupression and intense chemotherapy.[1]

> "Radiation therapy involving the head and neck region can result in temporary or permanent destruction of salivary glands."

Oral infections caused by HSV may be preceded by prodromal symptoms of pain, burning, tingling or itching.[6] They often begin as multiple vesicles at the vermillion borders, which rupture leaving painful ulcers that subsequently become encrusted with dried exudate.[6] HSV infections often follow an atypical course in immunocompromised patients.[35] Lesions are often larger and require up to four weeks or even longer for complete healing without antiviral therapy.[35]

Diagnostic tests include immuno-fluorescence, Tzanck test, polymerase chain reaction, antibody titres, Western Blot analysis, and electron microscopy. However, viral culturing offers the most sensitive diagnostic test for the detection of HSV infections.[35]

As a result of the increased susceptibility to HSV infections, the high frequency of atypical appearance and the prolonged clinical course of HSV infections, some investigators recommend prophylactic antiviral agents for patients who are severely myeloimmunosuppressed.[36,37] Acyclovir is the preferred treatment both prophylactically and therapeutically in sero-positive patients to prevent and control HSV infection.[5]

4. Haemorrhage

Cytotoxic chemotherapeutic agents may secondarily induce thrombocytopenia which is the usual cause of intraoral haemorrhage.[1,13,38] The haemorrhage may occur anywhere in the mouth and may occur spontaneously or secondary to trauma or inflammation.[13] Oral petechiae occur most often in patients with a platelet count below 50 x 10^9/L, while spontaneous haemorrhage is associated with platelet counts of less than 20 x 10^9/L.[3,13]

Prevention is the most effective technique used to avoid haemorrhage.[13] Eliminating potential areas of trauma (sharp restorations, fractured teeth, mobile deciduous teeth) and pre-existing intraoral disease before chemotherapy minimises haemorrhage.[13]

The oral mucosa should be kept moist with saline rinses, to avoid friction that may initiate bleeding.[13] Management requires removal of accumulated blood to identify the bleeding site and then pressure should be applied with moist gauze, periodontal packing or a mucosal guard. Management of haemorrhage may require antihaemorrhagic agents such as topical thrombin, absorbable gelatin sponges, oxidised cellulose, aminocaproic acid and microfibrillar collagen haemostat. If the bleeding episode is associated with an infection requiring surgical intervention or if local measures are unsuccessful, platelet transfusion may be required.[1,13]

Oral Complications of Radiation Therapy in the Head and Neck

The major early side effects of head and neck irradiation are mucositis, salivary gland changes, candidiasis and dysphagia.[5,18] Among post-irradiation problems are radiation-associated dental caries, periodontal deterioration, xerostomia, reduced maxillomandibular opening, decreased resiliency of perioral tissues and intrinsic bone changes.[5,18,39] Abnormalities in the developing dentition include; enamel hypoplasia, diminutive teeth, delay or failure of tooth development and eruption and altered root formation.[1]

Early Effects

1. Salivary Changes

Radiation therapy involving the head and neck region can result in temporary or permanent destruction of salivary glands.[1] An initial decrease in free flowing saliva is

Continued on Page 8...

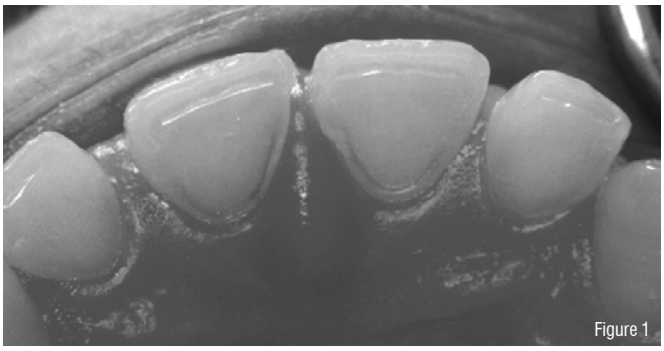


Figure 1

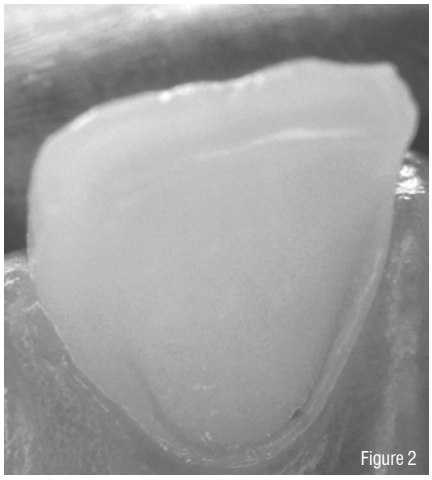


Figure 2

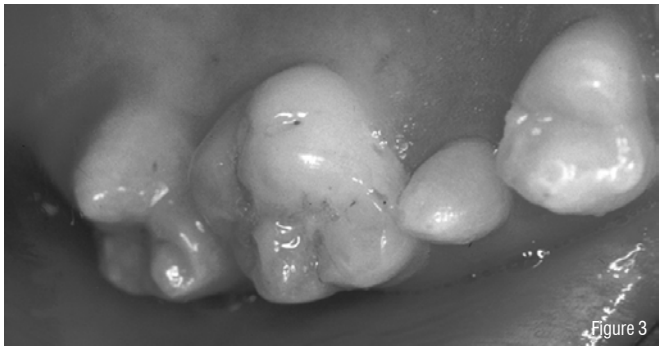


Figure 3

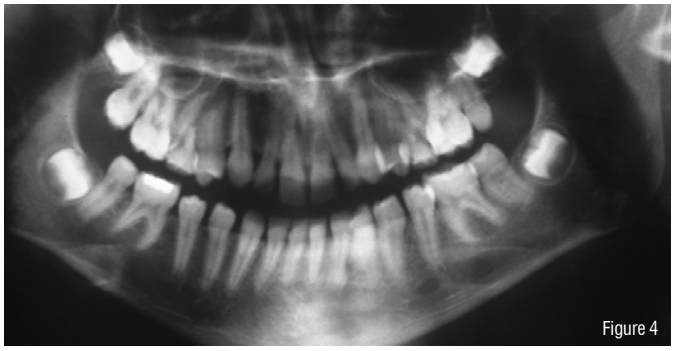


Figure 4

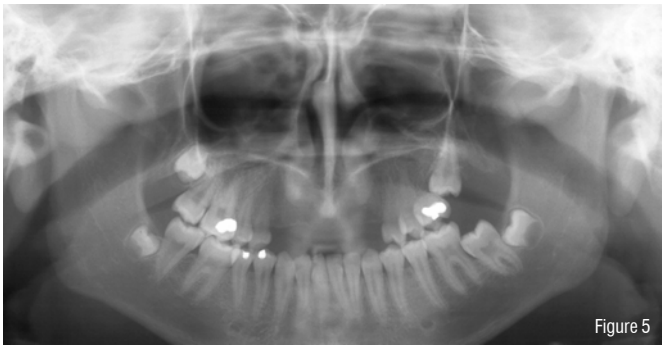


Figure 5

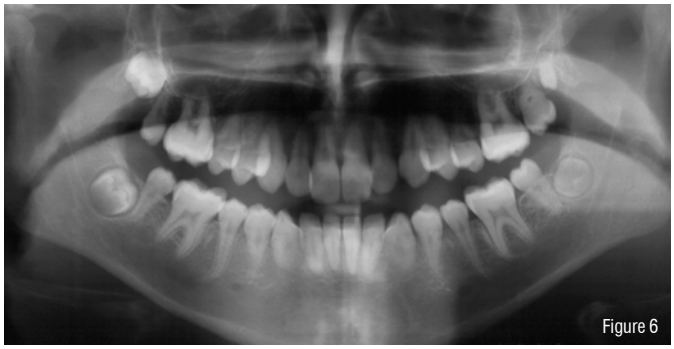


Figure 6

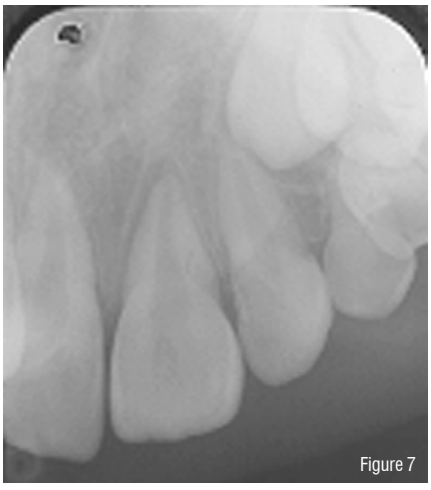


Figure 7

...Continued from Page 7

followed by an accumulation of sticky mucous, because the acini of serous glands are affected more than the acini of mucous glands.[5] The pH of affected saliva may fall to below 5.5.[5] The severity of salivary changes is related to the radiation dosage, quantity of salivary tissue exposed and the patient's age.[1,5] Altered salivary gland function predisposes such patients to pain, inflammation, dysphagia, xerostomia and severe radiation-associated dental caries.[1]

Managing xerostomia involves a combination of strategies including synthetic salivary substitutes, stimulation of remaining salivary tissue, maintenance of good oral hygiene and use of topical fluoride.[1] Patients with xerostomia whose salivary glands can respond to stimulation may benefit from using simple dietary measures such as eating carrots or celery or chewing sugarless gums.[13] Pilocarpine also appears to be effective in patients with residual salivary gland function, however patients with no functioning salivary glands require saliva substitutes.[13]

2. Mucositis

Mucositis induced by irradiation is defined as the reactive inflammatory-like process of the oropharyngeal mucous membrane following therapeutic irradiation of patients who have head and neck cancer.[40] It is important to record the extent of mucositis due to irradiation from both a clinical and research standpoint. A scoring system of mucositis enables communication between clinicians with respect to generalised complaints such as pain and dysphagia, as well documenting the dose-response relation for that patient.[40]

The first signs of mucositis, a whitish appearing mucosa, occurs after exposure to 1, 000 rads (10 Gy).[5,41] Eventually, the mucosal surface becomes erythematous with deposits of fibrin on the surface. In the uncontrolled patient, islands of ulceration may appear after 3, 000 rads (30 Gy).[5,41] As treatment progresses, the entire mucosa may be involved and the patient loses taste sensation. Local factors such as poor oral hygiene, dentures and defective teeth frequently aggravate the condition.[5,41,42]

3. Other Early Effects

Other acute complications of radiotherapy include loss of taste, dysphagia, trismus, loss of appetite, nausea, malaise and weight loss.[5] Patients who have received oral or radical neck surgery may also develop oedema of the buccal mucosa and floor of the mouth as a result of impaired venous and lymphatic drainage.[5]

Late Effects

1. Radiation Caries and Dental Erosion

A distinct type of rampant caries often occurs in patients receiving radiotherapy regardless of whether or not teeth are located within the collimation of the beam.[41,43] A reduction in the salivary flow together with a shift in the oral microflora towards more cariogenic organisms increases the risk of caries.[43]

Radiation caries usually appears in the cervical areas of teeth, if uncontrolled, caries will circle the entire tooth with subsequent crown fracture at the gingival margin.[5,43] Caries is also observed on the incisal edges of anterior teeth and the cusp tips of the posterior teeth, which are normally bathed in saliva and caries resistant.[5,43]

A major side effect of cancer treatment is nausea and vomiting. The reduced salivary flow in patients who have received head and neck irradiation renders the tooth surfaces particularly vulnerable to dental erosion. Erosion of the palatal surfaces of the upper anterior teeth are most frequently affected as shown in Figures 1 and 2.

2. Bone Changes and Developmental Defects

The most serious complication of radiotherapy is osteoradionecrosis.[43] Bone absorbs a larger amount of radiation compared to an equal volume of soft tissue.[5] Radiation reduces the number of bone cells as a result of endosteum atrophy and direct cell death of osteoblasts and osteocytes.[5,44] The bone becomes significantly acellular and avascular, with marrow spaces undergoing fibrosis and fatty degeneration.[5,44]

These changes make the bone vulnerable to trauma and infection impairing its capacity to remodel and repair.[43] The mandible's high bone density and low vascularity makes it more vulnerable to osteoradionecrosis than the maxilla. If growth centres are affected by irradiation, the development of the facial bones may give rise to facial asymmetry and malocclusion.[43]

The most common dental factors precipitating necrosis are pre- and post-radiation extractions and pre-existing periodontal disease.[43] Dentures that cause ulceration to the underlying mucosa and underlying bone are also contributing factors.[43]

The use of megavoltage radiation has reduced the incidence of osteoradionecrosis from around 37 percent to below 20 percent.[45] However, clinicians should perform extractions carefully on patients who have received radiotherapy.[5] In susceptible patients the correct extraction technique involves alveolectomy and primary closure of the socket to eliminate the potential for sharp bony edges or spicules that may damage the overlying mucosa.[5,46] This is performed under the cover of broad spectrum antibiotics.[5,46]

Wound healing of heavily irradiated bone following surgery can be enhanced with the use of hyperbaric oxygen.[39] Hyperbaric oxygen treatment requires about four weeks and results in revascularization of irradiated tissue.[39] The major difficulties of this treatment are time and expense. Soft tissue oxygen partial pressure (tension) can be raised to approximately 80 percent of normal after a total of twenty, one and a half hour treatments at 2.4 atmospheres of 100 percent oxygen.[39]

> "Children are particularly susceptible to both the acute and chronic oral manifestations of cancer treatment. Multidisciplinary treatment and coordination of care is therefore essential."

Radiotherapy may also damage developing tooth buds.[5,43,44,47] If irradiated before calcification is complete, the tooth buds may be destroyed.[44] Exposure later in the developmental process may arrest growth and cause irregularities in enamel and dentine.[44] Consequently these patients may have missing teeth, enamel opacities, hypocalcifications, thin or shortened roots and developmental malocclusions.[43,44] Interestingly, teeth often fully erupt even if the roots may fail to form.[44]

Figures 3 and 4 illustrate the same patient with hypoplasia of the second premolars and second molars. The OPG shown in Figure 5 presents a patient who received irradiation for treatment of a nasopharyngeal carcinoma. Note failure of tooth development in the anterior maxilla and impaction of the upper left second molar.

The reduction in root length may result in endodontic infections secondary to minimal periodontal breakdown.[43] Although it is not reported in the dental literature, such teeth may also be predisposed to luxation injuries as a result of the reduced periodontal attachment. Figure 6 is an OPG of a patient illustrating reduced root length of multiple teeth secondary to chemotherapy. This patient sustained a palatal luxation injury of tooth 21 as shown in Figure 7.

3. Other Late Effects

The effect of radiotherapy predisposes the periodontium to disease since both vascularity and the capacity of the supporting bone to remodel and repair is also reduced.[43] In addition, a reduction in the salivary flow increases plaque deposition. Teeth with advanced periodontitis are also at risk of initiating osteoradionecrosis through the invasion of microbes directly into the underlying bone.[43]

Candidiasis is also more common following radiotherapy which has resulted in xerostomia. Altered salivary flow and salivary histatin levels may be important predisposing factors. The increased susceptibility to candidiasis is particularly evident in patients wearing dentures.[43]

Trismus is another chronic complication of radiotherapy. A reduction in blood flow to the muscles of mastication as a result of progressive endarteritis results in scarring and fibrosis of the affected muscles.[43]

As treatment for paediatric cancers improves, the increased survival of these patients may result in an increase in the number of second primary malignancies. Van der Waal reports on a case of oral squamous cell carcinoma following treatment of acute lymphoblastic leukaemia.[48] It is tempting to speculate that the previous oncology treatment, either irradiation or cytotoxic chemotherapy, was related to this second neoplasm rather than a random event.[48]

In a retrospective study of 9720 children treated for acute lymphoblastic leukaemia, Neglia et. al., found a seven-fold excess of all cancers and a 22-fold excess of central nervous system tumours when compared to prevalence rates in the general population.[49] Children who underwent cranial irradiation before five years of age had the greatest susceptibility to brain tumours.[49]

Conclusion

Children are particularly susceptible to both the acute and chronic oral manifestations of cancer treatment. Multidisciplinary treatment and coordination of care is therefore essential to reduce both the incidence and morbidity of these complications.The dentist involved in the patients care must recognize the oral complications of cancer therapy which may affect cells either directly or secondarily through myeloimmunosuppression.

Please contact the Editor by email for references

BRANCH REPORTS

New South Wales

President: Eduardo A Alcaino
Secretary: Juliette Scott
Treasurer: Anthony Burges
Committee Members:
Joanna Seppelt, Suzanne Turek, Richard Widmer, Angus Cameron, Kareen Mekertichian and Mary Moss

There are approximately 50 active members of the Society for 2000. During this year, the society held three meetings at the usual venue, The Duxton Hotel at Milsons Point. Although 4 meetings are planned for each year, the 12th Biennial ANZSPD Conference in Adelaide (between the 24th-26th February 2000) also filled our calendar, with several of our members attending to this meeting. The three meetings for the year 2000 were as follows:

Tuesday 11th April 2000

This meeting discussed "Contemporary diet & nutrition and its effects on dental disease". Our guest speaker was A/Professor Linda Tapsell, Acting Director- Smart Foods Centre, The University of Wollongong. A/Prof. Tapsell spoke about current aspects of dietary advise, the effects of high frequency eating (snacking) in children and dental caries, erosive wear, and current protocols for pre-school children.

Of interest also was the 5th Congress of the European Academy of Paediatric Dentistry held from June 7-11 in Bergen, Norway. This was attended by several members of our state branch, with papers being presented by A/Prof R. Widmer, Dr A. Cameron, Dr E. Alcaino, and two of our post-graduate students, Dr E. Mahoney and Dr Katherine Ngu. This was a highly successful meeting with approximately 350 delegates attending.

Tuesday 1st August 2000

This meeting discussed "Pain management in the paediatric patient". Our guest speaker was Dr Suellen Walker, a staff specialist in the Department of Anaesthesia and Pain Management and Clinical Lecturer in the University of Sydney Pain Management and Research Centre, Royal North Shore Hospital. Dr Walker addressed current trends in pain management in the paediatric patient; she also discussed hierarchy of pharmacological management, and also focussed in pain management for the paediatric dental patient. This was an excellent talk with much interest from the audience.

Tuesday 7th November 2000

This event has been scheduled for the afternoon of Melbourne Cup Day. A short general trivia Quiz and Luncheon has been arranged for the afternoon. We will then join Melbourne Cup festivities, including a live coverage of the Melbourne Cup race. The afternoon is meant to be a social, informal and pleasurable event for all dentists and their staff.

Eduardo A. Alcaino
Hon Secretary ANZSPD 2000 (NSW Branch)

New Zealand

The New Zealand Branch of ANZSPD met in October at the NZDA biennial Conference. There we thanked some very long serving members being Mary Livingstone as Secretary/Treasurer and Alan Issac as committee member and previous president. The President is now Jo Pedlow from Christchurch, President Elect is Callum Durwood (Auckland) & Secretary Mary Anne Costelloe (Stratford, Taranaki). New Committee members are Nina Vassan (Auckland) and Christine Holloway (Timaru).

The conference enjoyed a good paediatric program (organised by Dr Bernadette Drummond). Angus Cameron was a key speaker and was very well received. Recent Otago Masters graduates Allison Meldrum and Katie Ayers were also part of an interesting and well attended program along with Dorothy Boyd

The society executive has convened regularly by teleconference throughout the last two years. Here the branch's constitution has been reviewed and updated with one notable requirement of the NZ registrar of Societies, that we must have an annual general meeting, being addressed. As a result, the society is now an incorporated body (many thanks to Mary Livingstone).

Submissions were also reviewed and forwarded to NZDA on several issues including competency and continuing education, along with a short submission on registration requirements for foreign graduates.

Auckland members ran a successful one day course with Drs Callum Durwood, Heather Keall, Kahtan Hameed, and Nina Vassan. being speakers.

Fluoridation has been an issue again in various centres. In New Plymouth the District Council opted to review the matter by having a public forum which lasted one day, rather than a referendum or a series of public meetings. This enabled interested parties to make submissions without interjections, and in a controlled environment. Submissions were varied & came from a wide range of the community including the youth council, Public Health officials. various health workers, the Pure Water association, and individuals wanting to control the poisons in their drinking water and their right to choose. Several local Maori groups spoke strongly in favour of retaining fluoride. Prior to the forum most councillors had indicated they wished to remove fluoride, but the decision was strongly in favour of continued fluoridation. Only one councillor out of the twelve voted against continued fluoridation

Next year the society is planning to have several visiting paediatric speakers including the ANSPD President Dr Kerrod Hallet, but first we are all anticipating family time over Christmas and a restful, peaceful summer holiday which we hope our Australian members will also enjoy.

Mary Anne Costelloe

Queensland

The year is coming to a close for the Qld Branch with our Annual Clinic Day and AGM in November. Under the auspices of our President, Dr. John Keys, the Branch

BRANCH REPORTS

has had excellent attendances for meetings this year.

Our meeting program commenced in February with Dr. Eric Hewett, an Anaesthetist, speaking on the risks in general anaesthetic dentistry. Following on, Barry Kerr, a Psychologist, presented the April Meeting on "How to survive paediatric dentistry". Needless to say, attendances at this meeting hit an all-time high!

We were fortunate to acquire a visit from an overseas speaker for the June Meeting. Dr. Rocio Quinonez, a Clinical Fellow from the Department of Paediatric Dentistry at the University of North Carolina had just completed her studies and presented her thesis findings on early childhood caries. Dr. Arabelle Clayden, an Oral Pathologist, spoke at the August meeting on oral medicine for children.

The Branch members have elected the organising committee for the 13th ANZSPD Biennial Conference to be held in Brisbane in 2002. The date and venue have been set: 14-16th November 2002 at the Hilton Hotel in Brisbane. After Adelaide's huge success in organising this year's 12th Biennial Conference, Brisbane has a hard act to follow - but it is a challenge we will rise to!

The Annual Clinic Day on November 18th is titled "Kids Teeth - Let's Get It Right" and promises to be the highlight of the year. Dr. Peter Gregory and Dr. Gil Shearer, a Brisbane Endodontist, are the guest speakers and COLGATE is generously sponsoring the event.

The year 2000 has again been a fruitful year for the Qld Branch. Members have enjoyed a varied scientific program and the convivial atmosphere of the dinner meetings has encouraged all members to interact on a social and professional level in support of the Society.

Irma Rutar
Secretary/Treasurer, Qld Branch ANZSPD

Tasmania

The main news from Tasmania, is that our president Tasha Dodd has just had a baby girl. There can't be to many presidents who have a baby in their term of office!

Anwen Sophia Catherine Crane: 9 pounds 2 ounces !

The other news is concerning the dental act which is being drafted and will hit parliament soon. The new act will have Dentists, Dental therapists and dental hygienists covered in the one act which is good news and the new act will finally allow dental hygienists to practice in this state after many, {15+} years of lobbying by the ADA Tasmania.

Clinical Hint:

To make the acceptance of the RA nose piece easier for the little ones. I sometimes draw "freckles" on it with a red felt pen. The hint is don't use permanent marker - use the whiteboard wipe off stuff otherwise the red dots are there for long time!

Wayne

Victoria

The Victorian Branch has had an active year, with five dinner meetings held at University House, which have been well attended by members, guests and dental therapists. Guest lecturers and topics were from a diverse range of disciplines. Our paediatric dentistry postgraduate students have additionally presented an interesting series of case reports.

At the initial February meeting Orthodontist, Dr Jeff Lipschatz presented a well received talk on "Orthodontic Impactions: the good, the bad, and the ugly."

At the April meeting Paedodontic postgraduate Dr Wina Darwis presented an informative outline of "Communication skills in managing children with cerebral palsy". Our guest speaker, well known Endodontist, Dr Paul Abbott then delivered a talk on "Digital imaging versus conventional radiography in dentistry".

At the June meeting we were entertained by Periodontist Dr Eryn Agnew who gave a humorous and informative lecture entitled "Children, have you brushed your teeth?", which followed a postgraduate case report by Dr Lochana Ramalingam on "Posterior cross bite correction with a quad helix appliance".

Our August meeting saw Branch member and Director of the Oral Health Therapy program in Victoria, Dr Hanny Calache deliver a dissertation entitled "The role of the Oral Health Therapist in paediatric dentistry", including an update on the present status and likely future concerning dental therapists and dental hygienists. This followed a post graduate case report by Dr Preenan Suwatviroj of "an anterior crossbite correction on an autistic child carried out under general anesthesia".

The Elsdon Storey Memorial lecture was delivered at the final dinner meeting in October by Dr Nicky Kilpatrick, Director, Department of Dentistry Royal Children's Hospital of Melbourne. Professor Storey's wife, Dr Pat Gladwell, gave a brief outline concerning Professor Storey's early dental research activities, publications, promotion and development of dental under graduate and postgraduate education in an era characterised by a dearth of scholarly dental activity, and of his continuing promotion of community water fluoridation.

The lecture entitled "Restore it, Extract it, Watch it or Refer it?" addressed the aetiology and management of the compromised hypoplastic and/or carious first permanent molar, this frequent dilemma facing clinicians today. This was preceded by a postgraduate case presentation by Dr Kylie Pearce entitled "Amelogenesis Imperfecta: from pedigree to practice".

Branch members remained for the Branch Annual General Meeting, which followed the evenings educational activities. The Branch's social end of year gathering is to be held on Friday 1st December at the home of Dr James and Isabelle Lucas in Malvern.

An innovative and stimulating program for 2001 has been developed by the Branch Executive.

Chris Olsen

Coming Events

- 7th World Congress on Preventive Dentistry. "Prevention in the 21st Century." Beijing, China. 24-27 April, 2001. Secretariat Office National Committee for Oral Health
  38 Baishiqiao Road,
  Haidian. Beijing, 100081, China.
  Congress Web Site:
  http://www.ciccst.org.cn/wcpd

- Australasian Academy of Paediatric Dentistry. Pre-Congress Meeting. Brisbane, Australia. 3-4 May, 2001.

- 30th Australian Dental Congress. Brisbane, Australia. 4-8 May, 2001. Contact Congress Secretariat
  PO Box 1280 Milton, Qld 4064
  e-mail: ada2001@im.com.au

- 9th International Congress on Cleft Palate and Related Craniofacial Anomalies. Göteborg, Sweden, 24-28 June 2001. Contact Conference Secretariat,
  Congrex Göteborg AB, Box 5078,
  SE 402 22 GÖTEBORG, sweden.

- I.A.P.D. Congress, Paris, France. 13 - 15 September 2001.

- 89th FDI Annual World Dental Congress. Kuala Lumpur, Malaysia. 16-19 September, 2001.
  Contact Mr Paul Wilson,
  FDI World Dental Federation Congress & Exhibition,
  7 Carlisle Street,
  London, England W1V 5RG.

- 13th Australian and New Zealand Biennial Conference. Brisbane, Queensland. 3 - 5 October 2002.

- I.A.P.D. Congress. New Orleans, U.S.A. 16 - 19 October 2003.

AUSTRALIAN AND NEW ZEALAND SOCIETY OF PAEDIATRIC DENTISTRY

Federal President
Kerrod B Hallett
MDSc, MPH, FRACDS
Paediatric Dentist
Children's Oral Health Service
Royal Children's Hospital
Herston Road
HERSTON Q 4029

Federal Secretary Manager
Dr Alistair Devlin
57 Burroughs Road
Karrinyup WA 6018
AUSTRALIA

Branch Executives

Branch	President	Secretary	Federal Councillor
NSW	Eduardo Alcaino	Dr Juliette Scott	Dr Richard Widmer
Qld	Dr John Keys	Dr Irma Ruta	Dr Kerrod Hallett
Sa	Dr Candy Thomas	Dr Christine Adams	Dr Candy Thomas
Tas	Dr Ashwani Gupta	Dr Mala Anthony	Dr Natasha Dodd
Vic	Dr John Sheahan	Dr Felicity Wardlaw	Dr Chris Olsen
WA	Dr John Winters	Dr Alistair Devlin	Dr John Winters
NZ	Dr Ian Esson	Dr Mary Livingston	Dr Callum Durward

Editor Synopses
Dr John Winters

Correspondence Synopses
The Editor, Synopses
Suite 7, The Perth Surgicentre
38 Meadowvale Ave
South Perth WA 6151
AUSTRALIA

Printing and Distribution
Colgate Oral Care
Level 15, 345 George Street
Sydney NSW 2000
AUSTRALIA

Colgate

Mailing List

The mailing list for the distribution of Synopses is maintained by Dr John Winters on behalf of the Federal Secretary/Manager of ANZSPD. It is compiled from information supplied by the Branch Secretaries. If there are errors in your mailing details, please contact Dr Winters or your Branch Secretary. DO NOT contact Colgate for address correction.

Submissions

All text for inclusion in Synopses must be submitted to the editor on floppy disk, zip disk, CD, or by e-mail. Both PC and Mac formats are accepted. Media will not be returned. Address e-mail to winters@q-net.net.au. Please enclose your contact details and e-mail address with all submissions.

Deadline Next Issue

1st February, 2001.